



TITLE:

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A case with sudden onset of position-dependent hypoxemia caused by reopening of foramen ovale

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Running Title: Position-dependent hypoxemia

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We report a case of a 68-year-old woman with sudden onset of position-dependant hypoxemia after recovering from pulmonary embolism. Transesophageal echocardiography(TEE) and cardiac catheterization revealed the presence of patent foramen ovale (PFO) and the right-to-left shunting with no evidence of the right-to-left pressure gradient especially in the right lateral decubitous position. Surgical closure of the PFO was performed because the right-to-left shunting caused the position dependent severe hypoxemia. At operation, it was noted that the patient's enlarged aortic root pressed the heart inferiorly and compressed upper part of the right atrium. A large Eustachian valve, which is an embryonic remnant of the right valve of the sinus venosus, was observed in the right atrium. Preceding pulmonary embolism could lead to a temporal elevation of the right heart and pulmonary arterial pressures and it caused the reopening of the closed foramen ovale. To the best of our knowledge, this is the first case of reopening of the foramen ovale causing sudden onset of position-dependent hypoxemia.

KEYWORDS: Patent foramen ovale; Hypoxemia; Pulmonary embolism

Introduction

Several anatomical alterations are known to induce the right-to-left shunting through an interatrial defect such as ASD or PFO. Here, we report a case with sudden onset of position-dependent hypoxemia, which was caused by reopening of the foramen ovale after pulmonary embolism under the presence of a large Eustachian valve and position-dependent compression by the enlarged aortic root.

Case report

A 68-year-old woman was transferred to our institution with progressive shortness of breath specifically in the right lateral decubitus position. She had been in good health until she was admitted to another hospital for the treatment of lumbar hernia approximately 3 months before. On the 5th operation day, she noted dyspnea during her first walking for rehabilitation. Pulmonary embolism was detected with an enhanced CT scan, and ultrasonography of the lower extremities revealed the presence of deep vein thrombi in both legs. She had anticoagulant therapy and she was gradually recovered from shortness of breath and hypoxemia. However, she suffered from severe and another kind of hypoxemia on the 15th operation day. Her dyspnea was induced specially in the right lateral decubitus position with a resting room air pulse oximetry saturation of 70%, and was relieved in the erect or sitting position with a recovery of the saturation up to 99%. This postural worsening was not observed in the first hypoxemic event. Although anticoagulant therapy was continued for 3 months, postural hypoxemia was not alleviated and she was referred to our institution for further analysis. On physical examination, no murmur was audible on repeated auscultation. The blood pressure was 124/76 mmHg and heart rate was 81 beats/min, respiratory rate was 26/min. All findings of physical examination were unchanged by posture except for severe cyanosis in the right lateral decubitus position. Laboratory data was normal. Marked hypoxemia was noted in the right lateral decubitus position with the following arterial blood gas levels: inspired 2l/hr oxygen; PaO₂, 55.3mmHg; PaCO₂, 42.1mmHg; and PH 7.40 (PaO₂, 120.7mmHg; PaCO₂, 37.2 mmHg; and PH 7.43 in the sitting position). The percentages of the venous to arterial shunt calculated during breathing 100% oxygen were increased in the right lateral decubitus position (23.3% vs. 3.95%, right lateral decubitus position vs. sitting position). An electrocardiogram was normal. A chest X-ray demonstrated normal lung fields and protrusion of the right first arch in the heart figure. CT scans of the chest confirmed the absence of thrombi in pulmonary arteries and arteriovenous malformations. A transthoracic echocardiogram showed well contracting heart chambers with normal sizes, normal heart valves. Also, no elevation of the right heart and pulmonary artery pressures was found.

The presence of PFO was detected with TEE. The blood stream of the right-to-left shunting across the PFO from inferior vena cava was observed with contrast and colored-Doppler echocardiography in the right lateral decubitus position. Supine and left lateral decubitus position abated the right-to-left shunting (Fig.1). Cardiac catheterization was performed in both the right lateral decubitus and supine positions. There was no significant coronary artery narrowing. There was elevation of neither right heart pressure nor pulmonary artery pressure. The cardiac catheterization confirmed the presence of the PFO with no evidence of the right-to-left pressure gradient (Fig.2). Blood gas sampling was conducted in each position and the percentage of pulmonary to systemic shunt (Q_p/Q_s) was calculated. The right-to-left shunting was significantly increased in the right lateral decubitus position (0.68 vs. 0.92, right lateral decubitus position vs. supine position). Surgical closure of the PFO was performed because the right-to-left shunting caused the position dependent severe hypoxemia. At operation, the presence of the PFO was confirmed and it was noted that the patient's enlarged aortic root pressed the heart inferiorly and compressed upper part of the right atrium. A large Eustachian valve, which is an embryonic remnant of the right valve of the sinus venosus, was observed in the right atrium. This valve serves to conduct the blood stream from the inferior vena cava through the fossa ovalis into the left atrium in the embryonic life. After surgery, the patient became asymptomatic and hypoxemia induced by the right lateral decubitus position disappeared and repeated blood gas analysis revealed that the position-dependent hypoxemia was no longer present. After a normal postsurgical course, the patient left the intensive care unit on the 3rd day and was discharged on the 14th day.

Discussion

A PFO is a defect in the atrial septum that results from incomplete fusion of the septum primum to the septum secundum. The persistence of the PFO into adulthood is common (25-30% in the general population) and sometimes leads to several complications, including paradoxical embolism of thrombi as well as air, migraine and refractory hypoxemia. Normally, even if a potential channel between the atria remains, the higher left atrial pressure keeps the flap-like valve of the foramen ovale opposed to the septum secundum. Most cases of sustained right to left shunting through the PFO are seen under conditions where right atrial pressure exceeds that of the left, making the potentially PFO open. However, in our case the right-to-left shunting was observed in the absence of any demonstrable pressure gradient between the right and left atria. The right-to-left shunting despite normal right atrial pressure has been reported after right pneumonectomy or in association with venous embryonic remnants [1]. In these situations, an altered geometric relation between the caval veins and the atrial septum presumably accounts for flow-related rather than pressure-related shunting.

This case study documents a 68-year-old woman patient presenting with hypoxemia caused by a significant increase in the right-to-left shunting across the PFO in the right lateral decubitus position. The right-sided cardiac pressures were normal in all recumbent positions. Transesophageal echocardiography and operative findings revealed that the upper part of the right atrium was compressed by the enlarged aortic root, and contrast and colored Doppler echocardiography visualized the direct blood flow from the inferior caval vein to the PFO in the right lateral decubitus position. These findings were not observed in the supine and left lateral decubitus position. Furthermore, a large Eustachian valve was observed in the right atrium during open heart surgery. It is likely that compression by the enlarged aortic root and the presence of the Eustachian valve changed a sac-like structure of the right atrium into a tube-like one especially in the right lateral decubitus position. This structural change lost the function of the right atrium as a chamber and it conducted the direct blood stream into the PFO (Fig. 3). Remaining question in this patient is why the right-to-left shunting across the PFO suddenly occurred. Preceding pulmonary embolism appears to be the key to answer the question. It is presumable that pulmonary embolism led to a

temporal elevation of the right heart and pulmonary arterial pressures and it caused the reopening of the closed foramen ovale. After anticoagulant therapy with warfarin, we observed normalized right-sided cardiac pressures but PFO remained. This may explain the cause of sudden occurrence of hypoxemia in this patient. There are no reports documenting the reopening of the PFO in adulthood, but following study suggests it's possibility. Shanoudy et al. reported an increased prevalence of PFO in subjects with obstructive sleep apnea (OSAS) compared with the control group (69% vs. 17%)[2]. It may be explained by the enhanced right-sided cardiac pressures due to transient but frequent elevations of right-sided pressure during apnea which could cause the reopening of the closed foramen ovale[3].

To the best of our knowledge, this is the first case of reopening of the foramen ovale, which caused sudden onset of position-dependent hypoxemia.

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Figure Legends

Figure 1

Longitudinal transesophageal echocardiograms with colored-Doppler in the mid-upper esophagus at 63° showing the direct blood flow from the inferior caval vein to the PFO and upper part of the right atrium was compressed by enlarged aortic root in the right lateral decubitous position.

Figure 2

Pressure data on the right heart catheterization and trans-septal left-heart catheterization via the PFO.

There was no right-to-left pressure gradient between right atrium and left atrium.

SVC: superior vena cava, IVC: inferior vena cava, RA: right atrium, LA: left atrium, RV: right ventricle,

PA: pulmonary artery, PCW pulmonary capillary wedge

Figure 3

Schema of our case. Compression by the enlarged aortic root and the presence of the Eustachian valve changed a sac-like structure of the right atrium into a tube-like one especially in the right lateral decubitus position. Ao: aorta, Eust: Eustachian valve

Figure 1.

Supine position

Right lateral decubitous position

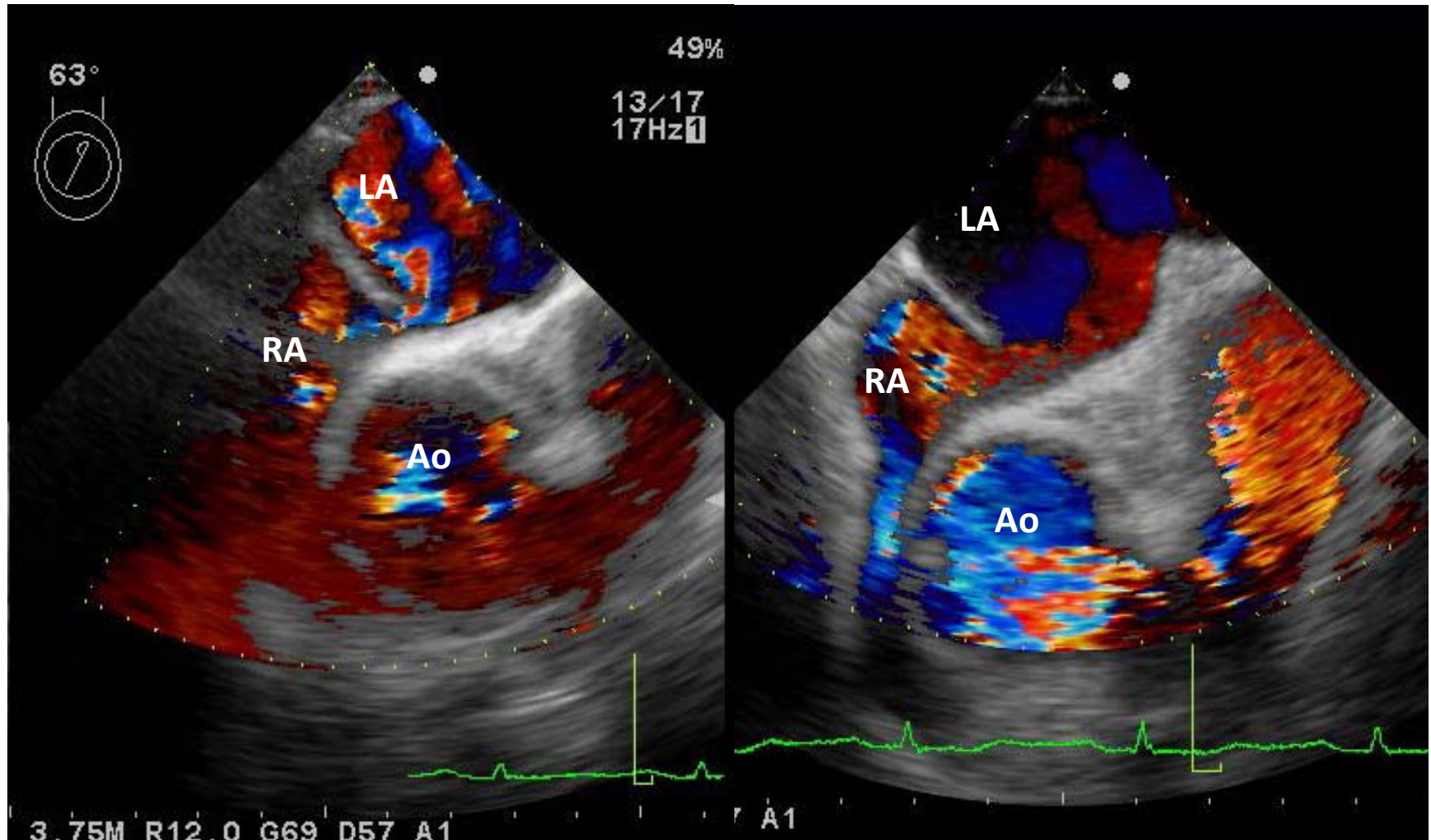


Figure2.



Locations	Supine position	Right lateral decubitus position
SVC	4/1/(2)	3/3/(3)
IVC	4/3/(3)	6/2/(3)
RA	5/3/(3)	4/0/(1)
LA	5/1/(3)	7/0/(2)
RV	21/1/(-3)	16/0/(-4)
PA	20/6/(12)	15/2/(7)
PCW	5/2/(2)	2/0/(1)

RA vs. LA pressures in the right lateral decubitus position

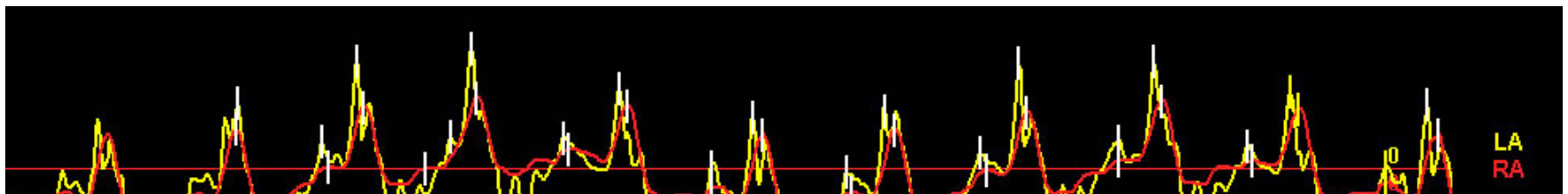
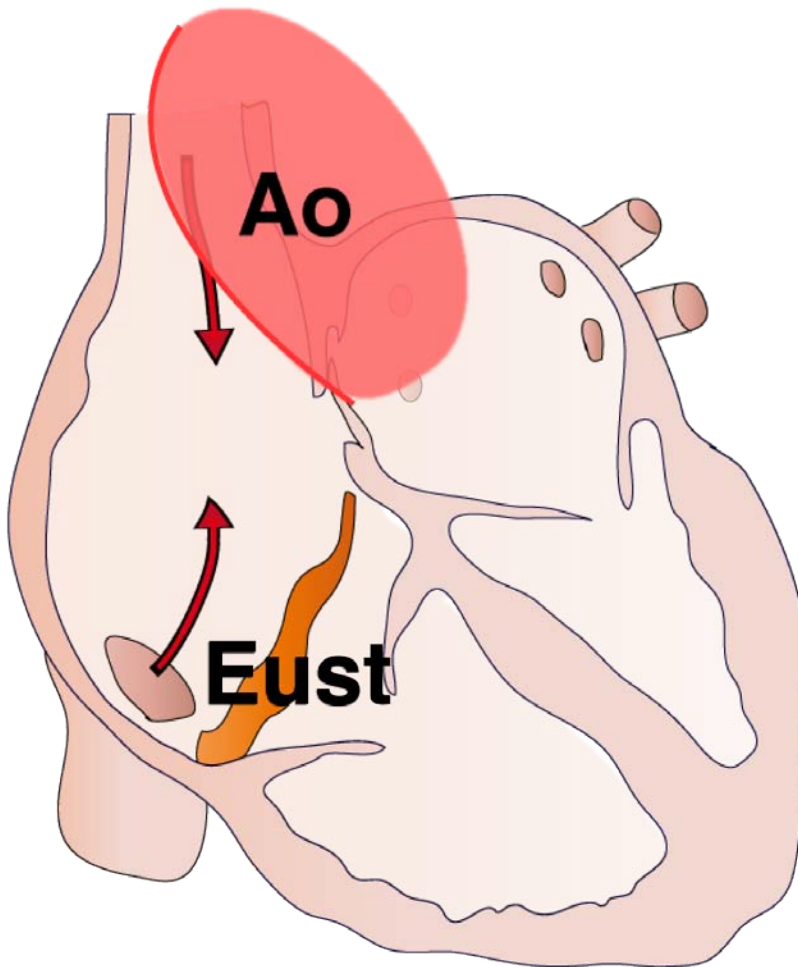


Figure 3.

Supine position



Right lateral decubitous position

